of adsorption the heat is all liberated in the short span of time during which the adsorbed layer is forming. It is therefore obvious that in spite of the fact that the heat of adsorption may be greater than that of condensation the rise in temperature observed would be mainly due to the oxidation of the alcohol as was found by experiment to be the case.

In view of what has preceded it is to be concluded that the rise in temperature of the platinum black due to adsorption of the alcohol vapor is not the initiator of the reactions which heat the platinum to incandescence. For the heat generated by adsorption is negligible compared to that produced by oxidation and would not be capable of increasing the speed of reaction appreciably.

It is then on the basis of an increased rate of oxidation of the alcoho at the surface of the platinum ("catalytic" action) coupled with the physical properties of the platinum black that one must explain the rise in temperature. In this, one is justified to some extent, for it has long been known that surfaces of platinum accelerate certain chemical reactions, both in gases and in solution. Recently, Langmuir¹ has shown that at ordinary temperatures and at reduced pressures platinum foil when "activated" will cause oxygen and carbon monoxide to unite. In this case there seems to be some close relation between this action of platinum and adsorption, but it is not due to any heating effect.

- <sup>1</sup> Langmuir, I., J. Amer. Chem. Soc., Easton, Pa., 40, 1918 (1361).
- <sup>2</sup> Zsigmondy, R., Zur Erkenntnis der Kolloide, Jena, 1905 (104).

## LEPTOSPIRA ICTEROIDES AND YELLOW FEVER

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Although yellow fever is being rapidly controlled by measures directed against the intermediate host (the Stegomyia mosquito), which acts as the vector, yet the nature of its inciting microbe has remained unknown. Hence yellow fever is a striking instance of the fact that given precise knowledge of the mode of infection of a microbic disease effective practical measures may be evolved for its control, even though the inciting microbe remain undiscovered.

Recently a fresh opportunity was afforded for the investigation of the etiology of yellow fever. The International Health Board of the Rockefeller Foundation sent a commission to Guayaquil, Ecuador, in June, 1918, to survey the field preparatory to the application of measures of control. Guayaquil has long been a recognized endemic focus of yellow fever, from which periodic extension has taken place to Central America.

I was attached to this commission as bacteriologist, and my attention was particularly directed, although not wholly confined to, the possibility that the inciting microbe belonged to the group of spiral microorganisms which in the last ten or twelve years have come to be regarded as playing a very important part in human pathology. There was already known, indeed, a particular spiral organism, *Leptospira icterohemorrhagiae*, which produces a severe and sometimes fatal jaundice in man, and which has a wide distribution in nature—far wider, indeed, than has yellow fever. This spiral microbe inhabits the rat, from which animal it finds its way at times to human beings, who then develop the symptoms and organic lesions of the disease called infectious jaundice.

I have given much attention to the spiral group of microorganisms for about ten years. In this period I have been enabled to devise new methods of cultivation, which have proven successful for species not before cultivated in successive generations, and which have also led to the discovery of new species. Hence in going to Guayaquil I took with me a laboratory outfit complete for this line of study.

Fortunately, I was successful in detecting in certain cases of yellow fever by culture methods and by guinea pig inoculation a particular spiral organism which I have since named *Leptospira icteroides*. The guinea pigs (and, as later studies showed, also puppies) successfully inoculated with the blood of yellow fever patients or with the cultures develop symptoms and lesions closely approximating those occurring in man and giving the clinical picture of the disease as usually observed. The outstanding signs are jaundice, hemorrhage into the lungs and stomach (this latter in man leads to the black vomit, so called), and albumin and casts in the urine. At autopsy, in the guinea pig as in man, the liver, kidneys, and other internal organs prove to be severely degenerated. The spiral organisms are, of course, recoverable from the inoculated guinea pigs, and with these organisms the disease is transmissible through an indefinite series of animals.

Moreover, guinea pigs have been successfully infected with the spiral organisms by means of Stegomyia mosquitoes, the vector in nature of the inciting microbe from man to man, and Stegomyias fed on infected guinea pigs are capable of transmitting the active microbe to still other guinea pigs, which, develop the symptoms and lesions described.

Finally, immunological studies have brought out important points of relationship (for example, to the Leptospira of infectious jaundice) and indicated the possibility of developing a "vaccine," and even a curative serum. But until the finding of *Leptospira icteroides* is confirmed by the investigation of cases of yellow fever in still other places, its standing as the inciting agent of yellow fever will have to be regarded as not yet certainly established.